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Term: (L7 or L6 or L5) and nitric oxide synthase

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#1 Search combrestatins	08:56:05	0

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Experimental Cell Research
Volume 218, Issue 1 , May 1995, Pages 189-200

doi:10.1006/excr.1995.1147 Cite or link using doi

Regular Article

Colchicine Induces Apoptosis in Cerebellar Granule Cells

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Available online 26 April 2002.

Abstract

Exposure to 1 &,micro;*M* colchicine, a microtubule disrupting agent, triggered apoptosis in rat cerebellar granule cells (CGC). Apoptotic nuclei began to appear after 12 h followed by oligonucleosomal DNA laddering, whereas inhibition of the mitochondrial 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazoliumbromide metabolism became significant between 18 and 24 h, when most cells already had apoptotic nuclei. These events were preceded by loss of tau protein and fragmentation of α and β tubulins. Colchicine treatment also caused alterations in Ca^{2+} responses to chemical depolarization and a moderate, but progressive, increase in the resting intracellular Ca^{2+} concentration. Nearly all neurons expressed c-Fos after the treatment with colchicine. However, while in part of the cell population c-Fos levels subsequently declined, in the neurons undergoing apoptosis the protein was still expressed, but had an abnormal intracellular localization. An increased expression of the constitutive nitric oxide synthase (NOS-I) was also detected at 12 h and was followed by increased nitrite production. Treatment with 100 n*M* taxol to stabilize the microtubuli prevented DNA laddering and apoptotic body formation induced by colchicine. In contrast, pretreatment with the *N*-methyl-D-aspartate receptor-antagonist, MK-801, or L-type Ca^{2+} channel blockers did not prevent colchicine-induced CGC

apoptosis. Inhibitors of NOS were also ineffective in preventing apoptotic body formation and DNA laddering, whereas they delayed the secondary cell lysis. These results support the idea that colchicine-induced cytoskeletal alterations directly initiate the genetic and structural modifications that result in CGC apoptosis.

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